BRONCHOGENIC CARCINOMA- CLINICAL, RADIOLOGICAL, PATHOLOGICAL CORRELATION

Dissertation submitted in partial fulfillment of regulation for the award of M.D. Degree in General Medicine (Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
March 2009

BRONCHOGENIC CARCINOMA- CLINICAL RADIOLOGICAL PATHOLOGICAL CORRELATION

Dissertation submitted in partial fulfillment of regulation for the award of M.D.

Degree in General Medicine (Branch I)

The Tamilnadu

Dr. M.G.R. Medical University

Chennai

March 2009

Coimbatore Medical College & Hospital

Coimbatore - 641 014

certificate

*This is to certify that the dissertation entitiled "**Bronchogenic Carcinoma- Clinical Radiological & Pathological Correlation"** , herewith submitted by **Dr Calwin Davidsingh. S**, post graduate in General Medicine Coimbatore Medical College Hospital is the record of a bonafide research work carried out by him under our guidance and supervision from July 2006 to June 2008.*

Prof Dr K.Umakanthan.MD
Unit chief & Head
Department of Medicine

Dean
Coimbatore Medical College
Coimbatore - 641 014

Date :
Place : Coimbatore

DECLARATION

I solemnly declare that the dissertation titled **"*Bronchogenic Carcinoma-Clinical Radiological & Pathological Correlation*"** was done by me at Coimbatore Medical College hospital from July 2006 to June 2008 under the guidance and supervision of **Prof Dr. UMAKANTHAN.K MD.** , Unit Chief and Head of Department**.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Dr. CALWIN DAVIDSINGH.S

Place : Coimbatore

Date : 01.12.2008

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The T.N. Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : Dr. Calwin David Singh

Course : M.D. (General Medicine)

Period of Study : 2006 - 2009

College : Coimbatore Medical College

Dissertation Topic : Bronchogenic Carcinoma - Clinical, Radiological And Pathological Correlation

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 5.3.2008

N. Thilakam

Secretary
Ethics Committee

ACKNOWLEDGEMENT

I express my sincere thanks to **Prof. Kumaran. MS.MCh.** Dean of Coimbatore Medical College Hospital for the permission to do this dissertation on Bronchogenic carcinoma. I am particularly thankful to my Unit Chief and Head of the Department **Prof Dr.Umakanthan.K MD** for the help, support and advice rendered in completion of my project. I wish to acknowledge the valuable guidance and encouragement given by **Prof Chitrakumar.MD,** Head of the Thoracic medicine Department in completing this dissertation. I am extremely grateful to **Dr. Vimala** Head of department of pathology for the assistance rendered throughout the project . I am also thankful to **Dr.Sudalaimuthu,** Head of Department of Radiology for the guidance in the project. I am extremely grateful to **Prof Dr.Yashodara** former Head of Department of medicine who helped and guided me in the study. I thank **Prof Neelambikai.MD,** Professor of Physiology and Head of Ethics Committee Coimbatore Medical College for the invaluable suggestions and corrections. I extend my special thanks to **Dr.Chandrasekar.S MD, Dr.Selvaraj MD, Dr.Neelakandan MD, Dr.Manohari MD and Dr.Avudaiappan MD** Asst Professors of Medicine for their assistance at various stages of completion of this project. I also express my sincere thanks to all my colleagues and juniors for their co operation. Finally I am grateful to all the patients who consented to take part in this study, for their valuable cooperation throughout the study.

CONTENTS

INTRODUCTION

Lung cancer is a major health problem worldwide. The incidence is increasing globally at a rate of 0.5% per year. The worldwide incidence is 14% whereas it constitutes 6.8% of all cancers in India. It is the leading cancer of both sexes in three of the Urban Cancer Registries (Bhopal, Delhi and Mumbai) in India. [1]

Unlike many other malignancies, whose causes are largely unknown, the cause of lung cancer is tobacco smoking in as many as 90% of patients. Although the relationship between cigarette smoking and squamous cell carcinoma and small cell carcinoma has long been clear, the relationship between smoking and adenocarcinoma, large cell carcinoma has been more ambiguous. While the older literature suggest that smoking is unrelated to adenocarcinoma more recent data indicates that all the major histopathological types are related to smoking.[2]

Adenocarcinoma is the most frequent Non Small Cell Cancer in the United States. Patterns of lung cancer in India varies from the western population. Squamous cell carcinoma is the commonest variety in india as compared to adenocarcinoma in the west.

The type and location of cancer influences the presentation in clinical practice. Central or endobronchial growth of the primary tumor may cause cough, hemoptysis, wheeze and stridor, dyspnea, and postobstructive pneumonitis . Peripheral growth of the primary tumor

may cause pain from pleural or chest wall involvement, cough, dyspnea on a restrictive basis, and symptoms of lung abscess resulting from tumor cavitation.

Although there are a plenty of studies relating smoking to Bronchogenic Carcinoma, there is a paucity of studies relating smoking to histopathological type. This study will focus on the various clinical, radiological presentations of Bronchogenic Carcinoma and its correlation to histopathological type.

AIM OF THE STUDY

1. To assess the relationship of histological type of Bronchogenic Carcinoma to smoking.

2. To study the clinical presentations of Bronchogenic Carcinoma.

3. To document the radiological appearances of Bronchogenic Carcinoma with histopathological correlation.

MATERIALS AND METHODS

Patients attending out patient department, Thoracic Medicine, Coimbatore Medical College Hospital, Coimbatore who were diagnosed to have Bronchogenic Carcinoma were selected.

Study Design:

Single Centre, Analytical Study

Study Period:

Study was conducted between January 2007 and August 2008 for a period of 18 months.

Sample Size:

In the study period of 18 months among patients attending Thoracic Medicine Out patient Department 50 patients were selected.

Selection Criteria:

All patients who had histological or cytological confirmation of lung cancer determined through bronchoscopy, CT scan guided FNAC/trucut biopsy or open biopsy are selected. Informed oral consent obtained from all patients. Detailed history, clinical Examination, Basic Biochemical Investigations were done in all patients.

REVIEW OF LITERATURE

SCENARIO OF LUNG CANCER IN INDIA

Lung cancer is the leading cause of cancer death in men and women in both the United States and the world. More Americans die each year of lung cancer than of colon, breast, and prostate cancer combined. The overall 5-year survival of lung cancer is 15% or less and has only shown minimal improvement over the past 30 years.

Lung cancer was initially thought to be infrequent in India. Lung cancer constituted 14.4% of all cancers. In a review of 9210 consecutive autopsies by Banker and Sirsat reported that lung cancer formed one per cent of all cancers in Tata Cancer Hospital. Viswanathan et al[25] collected information from different hospitals of the country and found that the incidence of lung cancer in hospital population was 27.4 per million in 1950 and in 78.6 per million in 1959. Lung cancer has remained predominantly a disease of males with a male to-female ratio ranging from 5.76:1 to 6.67:1. Around 80% of lung cancer patients come from the rural areas[7]. The demographic pattern of lung cancer in India is similar to that observed in Western countries 40 years ago. Forty per cent of patients of lung cancer are less than 50 years of age and 11% are less than 40 years.[7]

SMOKING AND LUNG CANCER

The vast majority (85–90%) of cases of lung cancer are attributable to smoking, and intensive research efforts have identified hundreds of carcinogens contained in both mainstream smoke (smoke directly inhaled by the smoker) and sidestream smoke (smoke released from burning tobacco between puffs plus smoke exhaled by the smoker). Although risk for lung cancer decreases significantly after smoking cessation, overall disease risk reduction takes years and an individual's risk never returns to that of a never smoker (never smoker is defined as fewer than 100 cigarettes in an individual's lifetime). Historically, 20 pack-years of tobacco exposure or more has been considered to contain the highest risk populations. Due to the large number of former smokers, new cases of lung cancer in the United States are diagnosed more commonly in former smokers than current smokers[7]. Smoking of bidi and hooka as well as cigarettes had similar Odds ratios for cumulative consumption. The risk increased with both the duration and quantity of all smoking products. Environmental tobacco smoke is a known lung carcinogen. Another study by Rapiti et al has shown that environmental tobacco smoke exposure during childhood is strongly associated with the risk of later development of lung cancer.

Other environmental factors can contribute to the development of lung cancer. Of particular note, many of these exposures, when coupled with smoking, lead to exponential increases in the risk of developing lung cancer. For example, smokers with asbestos exposure have a 50–100 times increased risk of developing lung cancer.

ENVIRONMENTAL RISK FACTORS FOR DEVELOPING LUNG CANCER

Proven

Passive/environmental tobacco smoke

Radon gas

Asbestos

Metals (chromium, arsenic, iron oxide)

Industrial (bischloromethyl ether)

Polycyclic aromatic hydrocarbons

Suspected

Air pollution

Vinyl chloride

Silica

History of tuberculosis

Genetics and lung cancer:

Cytogenetic studies have identified many chromosomal changes in lung cancer. These mutations include activation of the dominant cellular

protooncogenes of the ras and myc family and inactivation of the recessive or tumour suppressor genes. Small cell lung cancer is associated with oncogenes, like c-myc, L-myc, N-myc, c-raf and tumour suppressor genes, like p53 and Rb. Nonsmall cell lung cancer is associated with K-ras,N-ras, H-ras, c-myc, c-raf and tumour suppressor genes like p16 and Rb genes. FHIT is a tumour-suppressor gene and is frequently altered in lung cancer. Apoptosis or programmed cell death is altered in lung cancers due to changes in the anti-(BCL-3, Belxl) and proapoptotic members (Bax, Bad).

Diet And Lung Cancer:

There is some evidence that certain dietary factors may be protective for lung cancer, and others may increase the risk. There are conflicting reports about the role of betacarotene and lung cancer, although most reports suggest a protective effect.Sankaranarayanan found that green vegetables and bananas have a protective effect on the development of lung cancer. Pumpkins and onions had the most consistent protective effect. On the other hand, animal food products and diary products have a predisposing effect onlung cancer. Dietary cholesterol and animal fat increases the risk of lung cancer.

PATHOGENESIS:

Cancer develops in a multistep fashion in which cells become malignant by multiple genetic alterations affecting cellular growth,

differentiation, and survival. This can include the mutation of tumor suppressor genes (for example p53), the activation of oncogenes (for example, myc, jun, and fos), and the transformation of apoptotic genes. The overwhelming majority of cases of lung cancer are due to cigarette smoking. It is estimated that of cases of lung cancer, 90% in men and 80% in women are smoking related. Smoke contains hundreds of known carcinogens, including free radical oxidants and nonradical oxidants, which can damage DNA, proteins, and lipids. The chronic inflammation accompanying repeated smoke exposure also leads to genetic alterations in bronchial cells and contributes to development of lung cancer.

PATHOLOGICAL TYPES:

Small cell carcinoma:

This is a highly malignant tumour, with lymphocyte looking cells (twice the size of lymphocytes) – accounts for about 30% of lung cancer cases. They have little cytoplasm (i.e.: histologically basophilic). The cells grown in clusters. Electron microscopic studies show dense neurosecretory granules in some of these cells, and these granules are similar to those found in the neuroendocrine cells lining the bronchial epithelium. Also some cells exhibit presence of neuroendocrine markers such as: neuron-specific enolase and parathormone like active products. This suggests that these tumours have a origin from neuroendocrine cells lining the bronchial epithelium. Small cell carcinomas have strong

relationship to smoking, only 1% of non-smokers get it. They are usually located centrally in the hilar region. They metastasize rapidly, so surgical repair is not possible – chemotherapy is best. This sort of a tumour is commonly associated with a paraneoplastic syndrome (i.e.: especially if it produces ACTH & ADH).

Squamous Cell Carcinoma:

This accounts for about 45% of lung cancer cases. It is strongly correlated to a smoking history. It usually arises in the larger bronchi, more proximally. It is recognised by presence of individual cell keratinisation (keratin production causes cell death) and intercellular bridges. This type of cancer is commonly associated with: Pancoast tumour (i.e.: apical lung tumours in the superior pulmonary sulcus that tend to invade the neural structures around the trachea, cervical sympathetic plexus), hypercalcaemia. SCC is least likely to metastatize, at autopsy 50% of cases are confined to the thorax. This type of cancer may respond well to surgery.

Adenocarcinoma:

This accounts for about 20% of lung cancer cases. This is still associated with smoking, but is the most common type to occur in non-smokers & women. Histological classification includes two forms: 1) bronchial-derived adenocarcinoma (i.e.: derived from bronchial mucosa), 2) bronchioloalveolar carcinoma (i.e.: derived from terminal bronchioles

– specialised cells located here). Adenocarcinoma lesions are located more peripherally, are smaller, and vary histologically (glandular appearance → papillary lesions → solid mass). 80% of these tumours contain mucin producing glands.

Large Cell Carcinoma

This type of tumours probably represent SSC / adenocarcinoma, but because their cells are so undifferentiated – that they can no longer be recognised in the above category. Some of these tumour cells contain intracellular mucin, some cells are multinuclear (giant cell carcinoma), some have cleared cells (clear cell carcinoma), some have spindle shaped cells (spindle cell carcinoma).

CLINICAL PRESENTATION

The frequency of the common presenting symptoms of bronchial carcinoma

Symptom	**Frequency (%)**
Cough	41
Chest pain	22
Cough and pain	15
Hemoptysis	7
Chest infection	< 5
Malaise	< 5
Weight loss	< 5

Shortness of breath	< 5
Hoarseness	< 5

Symptoms due to local invasion:

The tumour may directly involve the pleura and ribs. Carcinoma in the apex of the lung can erode the ribs and involve the lower part of the brachial plexus (C8, T1 and T2), causing severe pain in the shoulder and down the inner surface of the arm (Pancoast's tumour). The sympathetic ganglion can also be involved, producing Horner's syndrome. Hilar tumours may involve the recurrent laryngeal nerve, causing unilateral vocal cord paresis with hoarseness and a bovine cough. Bronchial carcinoma can also directly invade the phrenic nerve, causing paralysis of the ipsilateral hemidiaphragm. It can involve the oesophagus, producing progressive dysphagia, and the pericardium, producing pericardial effusion and malignant dysrhythmias.

Superior vena caval obstruction causes early morning headache, facial congestion and oedema involving the upper limbs; the jugular veins are distended, as are the veins on the chest that form a collateral circulation with veins arising from the abdomen.

Metastatic complications:

Bony metastases are common, giving rise to severe pain and pathological fractures. There is frequent involvement of the liver.

Secondary deposits in the brain present as a change in personality, epilepsy or as a focal neurological lesion. Spinal cord compression is not uncommon and requires urgent treatment. Secondary deposits in the adrenal gland are a very frequent post-mortem finding but are often asymptomatic.

Non-metastatic extrapulmonary manifestations:

- ✓ Cachexia (anorexia, weight loss, weakness)
- ✓ Fever
- ✓ Hypertension

Endocrinological

- ✓ Hypercalcemia
- ✓ Hyponatremia
- ✓ Cushing's syndrome
- ✓ Gynecomastia
- ✓ Acromegaly
- ✓ Hypoglycemia

Neurological

- ✓ Lambert–Eaton myasthenic syndrome
- ✓ Peripheral neuropathy
- ✓ Cerebellar degeneration
- ✓ Limbic encephalitis

- ✓ Encephalomyelitis

Musculoskeletal

- ✓ Clubbing
- ✓ Hypertrophic pulmonary osteoarthropathy
- ✓ Dermatomyositis
- ✓ Polymyositis

Hematological

- ✓ Anemia
- ✓ Autoimmune hemolytic anemia
- ✓ Leukocytosis/thrombocytosis
- ✓ Vasculitis
- ✓ Noninfectious thrombotic endocarditis
- ✓ Idiopathic thrombocytopenic purpura

Hypertrophic pulmonary osteoarthropathy (HPOA) occurs in approximately 3% of all bronchial carcinomas, particularly squamous-cell carcinomas and adenocarcinomas. Symptoms include joint stiffness and severe pain in the wrists and ankles, sometimes associated with gynaecomastia. X-rays show a characteristic proliferative periostitis at the distal ends of long bones, which have an onion-skin appearance. HPOA is

invariably associated with clubbing of the fingers. It may regress after resection of the lung tumour or as a result of vagotomy at thoracotomy.

STAGING OF BRONCHOGENIC CARCINOMA

Stage	T	N	M	Description
0	Tis			Carcinoma in situ
IA	T1	N0	M0	Limited local disease without nodal or distant metastases
IB	T2	N0	M0	
IIA	T1	N1	M0	Limited local disease with ipsilateral hilar or peribronchial nodal involvement but not distant metastases or
IIB	T2	N1	M0	
	T3	N0	M0	Locally invasive disease without nodal or distant metastases
IIIA	T3	N1	M0	Locally invasive disease with ipsilateral or peribronchial nodal involvement but not distant metastases or
	T1–3	N2	M0	Limited or locally invasive disease with ipsilateral mediastinal or subcarinal nodal involvement but not distant metastases
IIIB	Any T	N3	M0	Any primary with contralateral mediastinal or hilar nodes, or ipsilateral scalene or supraclavicular nodes or
	T4	Any N	M0	Unresectable local invasion with any degree of adenopathy but no distant metatases; malignant pleural effusion
IV	Any T	Any N	M1	Distant metastases

Primary Tumor (T)

TX Primary tumor cannot be assessed; or tumor proved by the presence of malignant cells in sputum or bronchial washings but not visualized by imaging or bronchoscopy.

T0 No evidence of primary tumor.

Tis Carcinoma in situ.

T1 A tumor 3 cm in greatest dimension, surrounded by lung or visceral pleura, and without evidence of invasion proximal to a lobar bronchus at bronchoscopy.

T2 A tumor > 3.0 cm in greatest dimension, or a tumor of any size that either involves a main bronchus (but is 2 cm distal to the carina), invades the visceral pleura, or has associated atelectasis or obstructive pneumonitis extending to the hilar region. Any associated atelectasis or obstructive pneumonitis must involve less than an entire lung.

T3 A tumor of any size with direct extension into the chest wall (including superior sulcus tumors), the diaphragm, the mediastinal pleura, or the parietal pericardium; or a tumor in the main bronchus < 2 cm distal to the carina without involving the carina; or associated atelectasis or obstructive pneumonitis of the entire lung.

T4 A tumor of any size with invasion of the mediastinum, heart, great vessels, trachea, esophagus, vertebral body, or carina; or with a malignant

pleural or pericardial effusion; or with satellite tumor nodules within the ipsilateral lobe of the lung containing the primary tumor.

Regional Lymph Nodes (N)

NX Regional lymph nodes cannot be assessed.

N0 No demonstrable metastasis to regional lymph nodes.

N1 Metastasis to lymph nodes in the peribronchial or the ipsilateral hilar region, or both, including direct extension.

N2 Metastasis to ipsilateral mediastinal lymph nodes and/or subcarinal lymph nodes.

N3 Metastasis to contralateral mediastinal lymph nodes, contralateral hilar lymph nodes, ipsilateral or contralateral scalene or supraclavicular lymph nodes.

Distant Metastases (M)

MX Presence of distant metastasis cannot be assessed.

M0 No (known) distant metastasis.

M1 Distant metastasis present.

DIAGNOSIS OF BRONCHOGENIC CARCINOMA

The diagnosis of lung cancer rests on examination of a tissue or cytology specimen. Sputum cytology is highly specific but insensitive; the yield is highest when there are lesions in the central airways. Thoracentesis (sensitivity 50–65%) can be used to establish a diagnosis of lung cancer in patients with malignant pleural effusions. If cytologic examination of an adequate sample (50–100 mL) of pleural fluid is nondiagnostic, the procedure should be repeated once. If results remain negative, thoracoscopy is preferred to blind pleural biopsy. Fine-needle aspiration (FNA) of palpable supraclavicular or cervical lymph nodes is frequently diagnostic. Serum tumor markers are neither sensitive nor specific enough to aid in diagnosis.

Fiberoptic bronchoscopy allows visualization of the major airways, cytology brushing of visible lesions or lavage of lung segments with cytologic evaluation of specimens, direct biopsy of endobronchial abnormalities, blind transbronchial biopsy of the pulmonary parenchyma or peripheral nodules, and FNA biopsy of mediastinal lymph nodes. Diagnostic yield varies widely (10–90%) depending on the size of the lesion and its location. Recent advances include fluorescence bronchoscopy, which improves the ability to identify early endobronchial lesions; and endoscopic ultrasound, which permits more accurate direction of FNA. Transthoracic needle aspiration (TTNA) has a

sensitivity between 50% and 97%. Mediastinoscopy, video-assisted thoracoscopic surgery (VATS), and thoracotomy are necessary in cases where less invasive techniques fail to yield a diagnosis.

Part A: Recommended tests for all patients

- Complete blood count
- Electrolytes, calcium, alkaline phosphatase, albumin, AST, ALT, total bilirubin, creatinine
- Chest radiograph
- CT of chest through the adrenal glands.
- Pathologic confirmation of malignancy

Part B: Recommended tests for selected but not all patients:

MANAGEMENT OF LUNG CANCER

Surgery:

Surgery, radiotherapy and chemotherapy are the various options available for the management of lung cancer. In the early stages of NSCLC (Stage I to IIIA), surgery if feasible is the treatment of choice. The five-year survival rate after surgery are as follows: Stage I: 60-70%,State IA: (T1N0), 80%, Stage II: 35-40%, Stage IIIA (N2): 10-15%. As most cases of lung cancer present in an advanced and inoperable stage, and radiotherapy is only a local form of therapy, chemotherapy has

an important role in the management of lung cancer. Several regimens of chemotherapeutic agents have been studied in lung cancer. In a recent meta-analysis of randomized trials that compared chemotherapy with good supportive care, chemotherapy showed a modest benefit. There is an improvement in the quality of life, prolongation of median survival by 1.5-3 months, increased survival at one year by 10% and a reduction in the risk of death by 27 percent.

Chemotheraphy:

Chemotherapy is indicated for stage III B, stage IV non small cell carcinoma and all small cell carcinoma patients. Newer chemotherapeutic agents that have increased one-year survival up to 40% and median survival of about 8-9 months are being increasingly used now a days . These include Gemeitabine, Docetaxel, paclitaxel, Vinorelbine, Topotecan, Irinotecan, and Newer Platinum agents (carboplatin, Oxaloplatin, etc). Combinations of a platinum agent with a new generation cytotoxic agent have become the standard of care for first-line chemotherapy of advanced non-small cell lung cancer. In the presence of contraindications for platinumbased chemotherapy, platinum-free chemotherapy might be a reasonable option. There is not enough evidence to support the use of triple-drug chemotherapy. Administration of single-agent gemcitabine or vinorelbine can be considered in patients with poor performance status and in elderly patients. In case of

nonprogression and lack of severe toxicity, the administration of four to six cycles of chemotherapy is recommended. There is no evidence that prolongation of treatment has an impact upon survival. Second line chemotherapy using docetaxel should be considered for chemotherapeutically pre-treated patients with good performance status in order to relieve symptoms, prolong survival and improve quality of life. Studies from India have shown that without chemotherpay the median survival of unresectable NSCLC is five weeks, with a single agent it is 7.5 weeks, with less effective chemotherapy it is 9.5 weeks and with modern chemotherapy it is 23 weeks to more than 40 weeks. The problems with chemotherapy in India include a large number of dropouts, because of the costs and the side effects.

DATA ANALYSIS

TABLE 1

TABLE SHOWING SEX INCIDENCE

Table 1 shows that out of 45 patients 41(91%) were male and 4(9%) were female. The male to female ratio is 14:1.

TABLE 2

AGE INCIDENCE OF BRONCHOGENIC CARCINOMA

Age	Male	Female
41 to 50	4	0
51 to 60	23	3
61 to 70	18	0

From the above table it is found that 26 patients were within the age group 51 to 60 years which constitutes 57% of the patients studied. Only 4 patients were below 50 years and the rest 41 patients (91%) were above 50 years.

The mean age of incidence as per study is 59 years both in males and females.

TABLE 3

PREVALENCE OF SMOKING IN MALES AND FEMALES

	Male	**Female**
Smokers	40	1
Non Smokers	1	3

Among 45 patients studied 41(91%) were smokers and 4 were non smokers. Among 4 females studied 3 were non smokers and one had the habit of tobacco chewing.

TABLE 4

TABLE SHOWING DIFFERENT SMOKING PATTERNS

Among 42 patients who are smokers, 34 patients smoked bidi, 17 patients cigarette, 10 patients both bidi and cigarette and one patient had the habit of tobacco chewing.

TABLE 5

CORRELATION OF PACK YEARS AND BRONCHOGENIC CARCINOMA

Out of 41 patients who are smokers, 15 patients(36.5%) had smoking pack years less than 40 and 26 patients(53.4%) had pack years greater than 40. The maximum number of patients nearly 56% had smoking pack year between 41 to 80.

TABLE 6

CORRELATION OF TYPE OF BRONCHOGENIC CARCINOMA WITH SMOKING

Pathological type of tumor	Smokers	Non smokers
Squamous cell carcinoma	26	3
Adenocarcinoma	11	1
Small cell carcinoma	4	0

Out of 29 patients who had squamous cell carcinoma, 26 were smokers and 3 were non smokers. Among 12 patients who had adeno carcinoma 11 were smokers and one patient was a non smoker. The 4 patients found to have small cell carcinoma were smokers.

Among the 4 patients who were non smokers, 2 had adenocarcinoma and 2 had squamous cell carcinoma.

TABLE 7

TABLE SHOWING COMMON SYMPTOMS IN BRONCHOGENIC CARCINOMA

Symptoms	No of patients	Percent
Cough	44	97
Dyspnoea	26	57
Hemoptysis	23	51
Chest pain	20	44
Hiccough	3	6
Hoarseness of voice	2	4
Dysphagia	2	4
Brachial neuralgia	1	2

Out of 45 patients studied almost every patient had (97 %) cough as the predominant symptom. Dyspnoea is the next common presenting symptom which is present in 26 patients(57%). Hemoptysis is present in 51 % of patients and chest pain in 44 % of patients. The remaining symptoms found in small no of patients are hoarseness of voice, dysphagia, hiccough and brachial neuralgia.

TABLE 8

RELATIONSHIP BETWEEN SVC OBSTRUCTION AND PATHOLOGICAL TYPE

SVC obstruction	Squamous cell carcinoma	Adeno carcinoma	Small cell carcinoma
Present	2	12	0
Absent	27	0	4

Superior venacava obstruction is seen in 2 patients in our study and they both were found to have squamous cell carcinoma.

TABLE 9

RELATIONSHIP OF CLUBBING WITH PATHOLOGICAL TYPE OF BRONCHOGENIC CARCINOMA

Clubbing	Present	Absent
Squamous cell carcinoma	17	12
Adeno carcinoma	2	10
Small cell carcinoma	0	4

Clubbing was present in 19 patients in the study. Out of 19 patients, 17 patients(89%) had squamous cell carcinoma and the remaining 2 had adenocarcinoma.

TABLE 10

INCIDENCE OF PATHOLOGICAL TYPES OF BRONCHOGENIC CARCINOMA

Pathological type	No of patients	Percent
Squamous cell carcinoma	29	64
Adenocarcinoma	12	26
Small cell carcinoma	4	8

Among 45 patients with bronchogenic carcinoma 29 patients (64%) had squamous cell carcinoma, 12 patients (26%) had adenocarcinoma and 4 patients(8%) had small cell carcinoma.

TABLE 11

RELATIONSHIP BETWEEN PATHOLOGICAL TYPE AND SEX OF THE PATIENT

Pathological type	Male	Female
Squamous cell carcinoma	27	2
Adenocarcinoma	10	2
Small cell carcinoma	4	0

Out of 29 patients with squamous cell carcinoma 27 patients were males and 2 patients were females. Out of 12 patients with adenocarcinoma 10 were males and 2 were females. Among 4 patients with small cell carcinoma 4 were males.

TABLE 12

CORRELATION BETWEEN PATHOLOGICAL TYPE AND LOCATION OF TUMOR

Pathological type	Central	Peripheral	Total
Squamous cell carcinoma	20(69%)	9(31%)	29
Adeno carcinoma	3(25%)	9(75%)	12
Small cell carcinoma	4	0	4
Total	27(60%)	18(40%)	45

Among 29 patients with squamous cell carcinoma 20 patients(69%) had centrally located tumor and 9 patients(31%) had peripheral tumor. Among 12 patients with adenocarcinoma, 9 patients(75%) had peripheral tumor and 3 patients(25%) had central tumor. Among 4 patients with small cell carcinoma 4 had centrally located tumor.

TABLE 13

RELATIONSHIP BETWEEN THE RADIOLOGICAL PATTERN AND PATHOLOGICAL TYPE OF BRONCHOGENIC CARCINOMA

Radiological pattern	Squamous cell carcinoma	Adeno carcinoma	Small cell carcinoma
Mass lesion	23	5	3
Pleural effusion	3	3	1
Obstructive pneumonia	2	3	0
Mediastinal invasion	1	2	1
Rib erosion	5	5	1
Calcification	1	1	0
Cavitation	1	0	0

Out of 45 patients, 31 patients(69%) presented as mass lesion in the lung, 7 patients(15%) presented as pleural effusion, 5 patients(11%) presented as obstructive pneumonia.

Among 29 patients with squamous cell carcinoma 23 patients (79%) presented as mass lesion, followed by pleural effusion and obstructive pneumonia. Among 12 patients with adeno carcinoma 5

patients(41%) presented as mass lesion, 3 patients(25%) as pleural effusion and 3(25%) patients as obstructive pneumonia. In the small cell carcinoma three patients presented as mass lesion and one patient as obstructive pneumonia.

TABLE 14

CORRELATION BETWEEN RADIOLOGICAL PATTERN AND LOCATION OF LESION

Radiological pattern	Central	Peripheral
Mass lesion	22	9
Pleural effusion	2	5
Obstructive pneumonia	2	3
Mediastinal invasion	2	2
Rib erosion	4	7
Calcification	1	1
Cavitation	0	1

Among 45 patients with bronchogenic carcinoma 27 patients (60%) presented as a mass lesion, 7 patients (15%) presented as pleural effusion and 5 patients (11%) presented as obstructive pneumonia.

Among the central tumors, 22 patients (81%) presented as mass lesion, 2 patients(7%) as pleural effusion and 2 patients(7%) as obstructive pneumonia.

Among peripheral tumors 9 patients(50%) presented as mass lesion, 5 patients(27.7%) presented as pleural effusion and 3

patients(16.6%) presented as obstructive pneumonia.

Among 45 patients with bronchogenic carcinoma, 11 patients had rib invasion, 4 patients had mediastinal invasion, 2 patients had calcification and one patient had cavitation.

TABLE 15

TABLE SHOWING RELATIONSHIP BETWEEN

PATHOLOGICAL TYPES AND METASTASIS

Metastasis	Squamous cell carcinoma	Adeno carcinoma	Small cell carcinoma
Lymph node	8	1	0
Liver	4	1	1
Skeletal	0	1	0
Brain	4	2	1
Adrenal	1	1	0
Spleen	1	0	0
Intra pulmonary	1	0	0

9(20%) patients out of 45 patients had supraclavicular lymphnode metastasis. Six (13%) patients out of 45 patients had liver metastasis. Seven patients(15%) out of 45 had brain metastasis. 2 patients had adrenal, one patient had spleen and one had intrapulmonary secondaries.

TABLE 16

RELATIONSHIP BETWEEN THE PATHOLOGICAL TYPE AND LOBE INVOLVEMENT

Lobe	Squamous cell carcinoma	Adeno carcinoma	Small cell carcinoma	Total
Right upper lobe	8	6	1	15
Right middle lobe	3	0	0	3
Right lower lobe	7	4	1	12
Left upper lobe	9	2	1	12
Left lower lobe	2	0	1	3

Out of 45 patients, 15 patients (33%) had the lesion in right upper lobe, 3 patients (6%) had the lesion in right middle lobe, 12 patients(26.6%) had the lesion in right lower lobe.

Among 45 patients,12 patients (26.6%) had lesion in the left upper

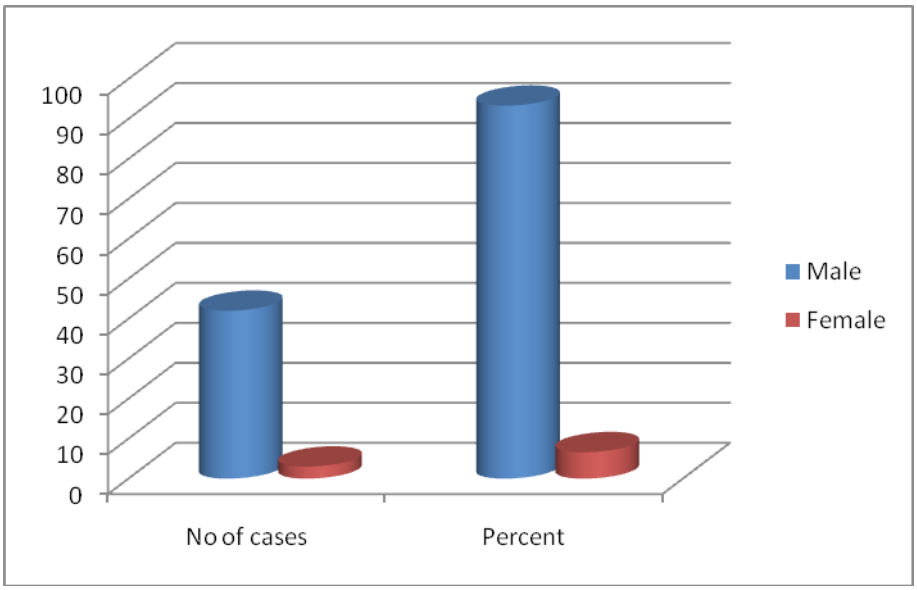
lobe and 3 patients (6%) had lesion in the left lower lobe.

Out of 45 patients, 30 patients(66.6%) had lesion in the right side and the remaining 15(33%) patients had the lesion in the left lobe.

Out of 45 patients, 30 patients had the lesion located in the upper lobe(including middle lobe) and 15 patients in the lower lobe.

DISCUSSION

SEX INCIDENCE

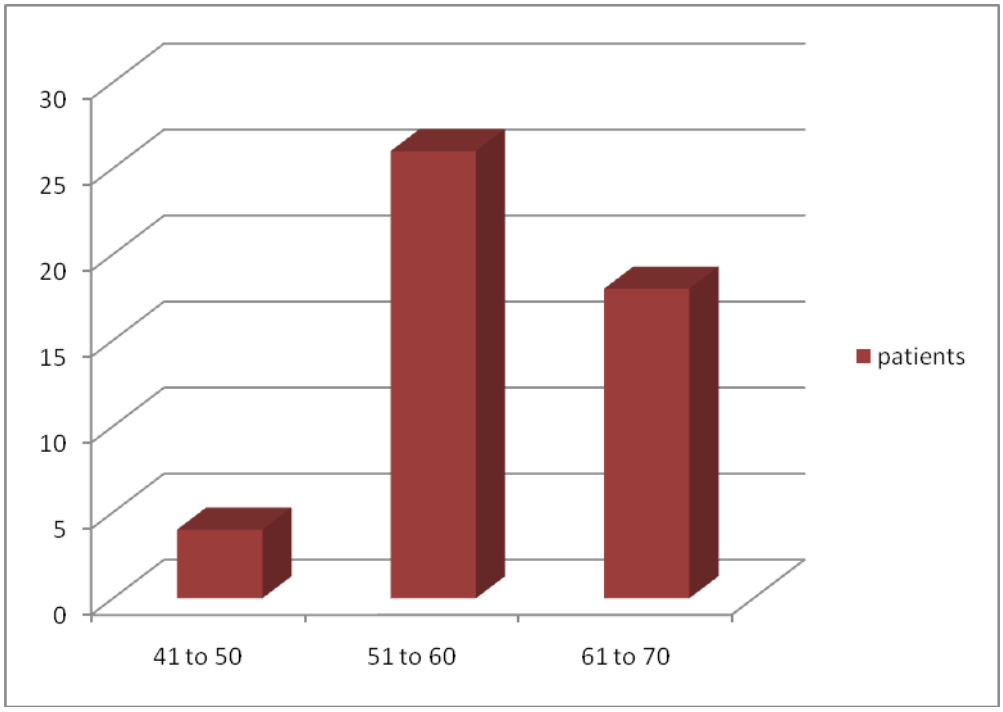


The male to female ratio in our study is 14: 1.

In a study conducted in northern india in Chandigarh in 2005[1], the overall male to female ratio is 5.2 : 1, which is much low when compared to our study. In a study conducted in Pakistan in the year 2002[2] the male to female ratio is 6: 1. In western countries the sex ratio is in the range of 3to 4: 1.

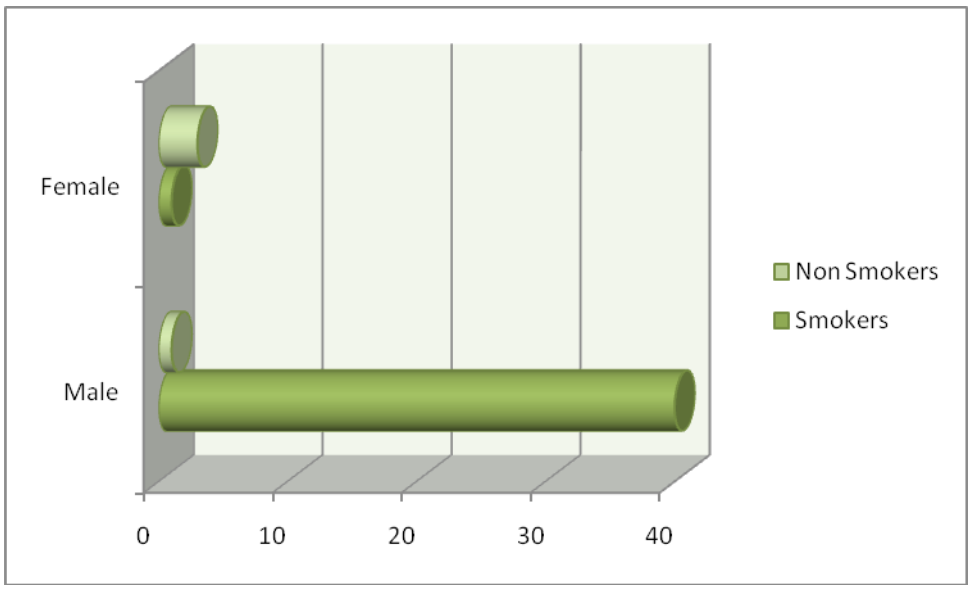
The recent increase in incidence among women in the West, which has been associated with a great increase in their smoking, may now give rise to the fall in sex ratio which Ochsner and De Bakey anticipated[44]. The low incidence in south Indians is attributed to less incidence of smoking in women.

AGE INCIDENCE OF BRONCHOGENIC CARCINOMA



The average age incidence in our study is 59 years, which is similar to the study conducted in Chandigarh in 2005[1]. In the study done in western Nepal[17] in 2001 the median age of the male and female patients was 67 and 66 years respectively. Increased smoking, urbanisation, and the introduction of new industries has probably led to a rise in the incidence of bronchogenic carcinoma in India at an earlier age.

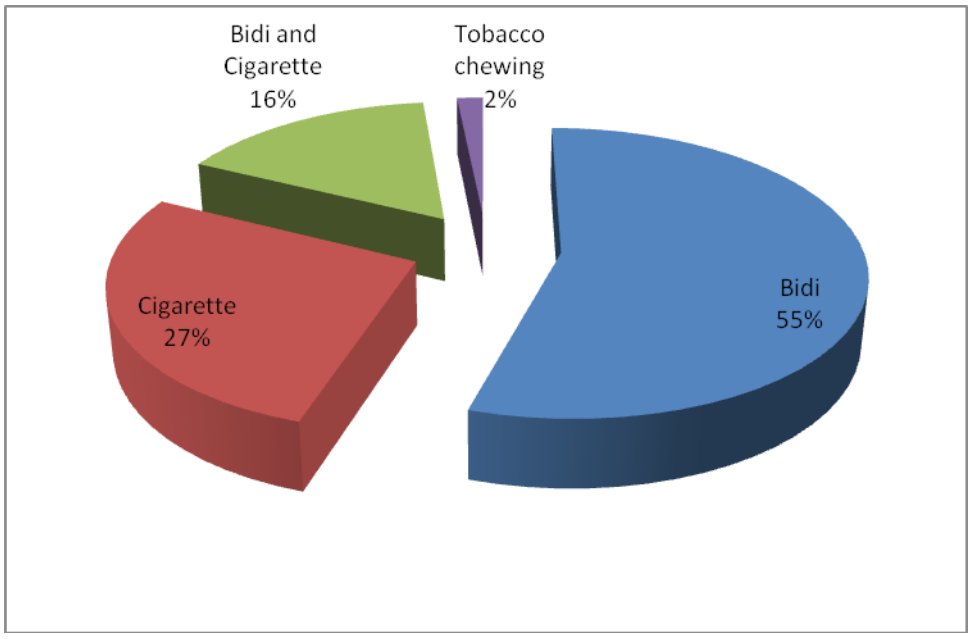
PREVALENCE OF SMOKING IN MALES AND FEMALES



In our study among 41 males 40 were smokers. Among 4 females one was a smoker. Smoking as a major risk factor in development of bronchogenic carcinoma among males **is statistically significant with p value <0.05.**

Our result correlates with the study in northern india In Chandigarh[1]. Our study also correlates with the study by CM shetty in Karnataka[3] where all non smokers were females. The less prevalence of smoking in females also attributes to less incidence of bronchogenic carcinoma among females. Smoking is a major risk factor for development of bronchogenic carcinoma in males. Other risk factors like occupational exposure to asbestos, arsenic, nickel, radiation, hereditary predisposition and passive smoking should be thought of in non smokers.

PREVALENCE OF DIFFERENT SMOKING PATTERNS



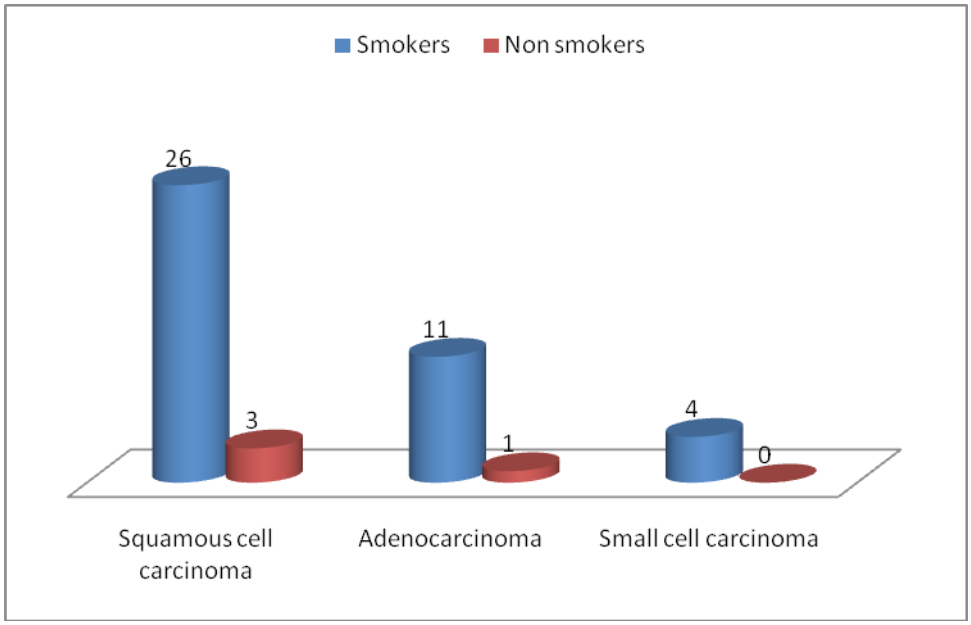
The most common pattern of smoking in our study is bidi (55%). Twenty seven percent had the habit of smoking cigarette. 16% had the habit of smoking both bidi and cigarette. One female had the habit of smoking tobacco.

In the study conducted in northern india[1], bidi smoking is the major pattern in Chandigarh patients affected by bronchogenic carcinoma. Bidi and cigarette scored the second place followed by cigarette alone as in our study.

In India the smoking of manufactured cigarettes is less common

than in Western countries. Not only do bidis contain less tobacco than the common types of Indian cigarettes but the tobacco is dried naturally and in this respect resembles cigar and pipe tobacco. Bidi smokers generally inhale deeply and Jindal and Malik[40] showed that as great a rise in carboxyhaemoglobin concentration may occur after smoking a bidi as after a cigarette. Possibly therefore the lower risk of carcinoma associated with pipes and cigars is due not to their containing naturally cured tobacco (as opposed to the flue-cured tobacco of cigarettes) but rather to the fact that pipe and cigar smokers generally do not inhale. Hence bidi smokers generally inhale deeply and have a greater risk of developing bronchogenic carcinoma when compared to cigarette smokers.

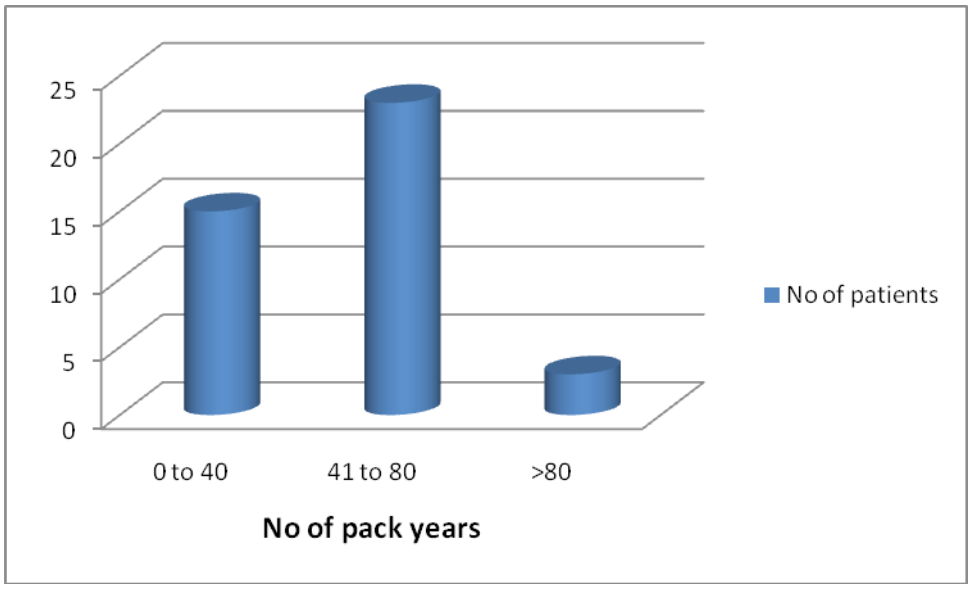
RELATIONSHIP BETWEEN PATHOLOGICAL TYPES AND SMOKING



In our study among 29 patients with squamous cell carcinoma 26(89%) were smokers and 3(11%) were non smokers. Among 12 patients with adenocarcinoma 11 were smokers. Among 4 patients with small cell carcinoma all were smokers. The role of tobacco smoke in the development of lung cancer is well known for squamous and small cell types, somewhat less so for adenocarcinoma. In a study conducted by Morabia A, Wynder EL[45], in USA smoking was associated with each lung cancer cell type, and differences in smoking habits by cell types were small.

Similar to the above said study, in our study all pathological types of bronchogenic carcinoma were associated with smoking.

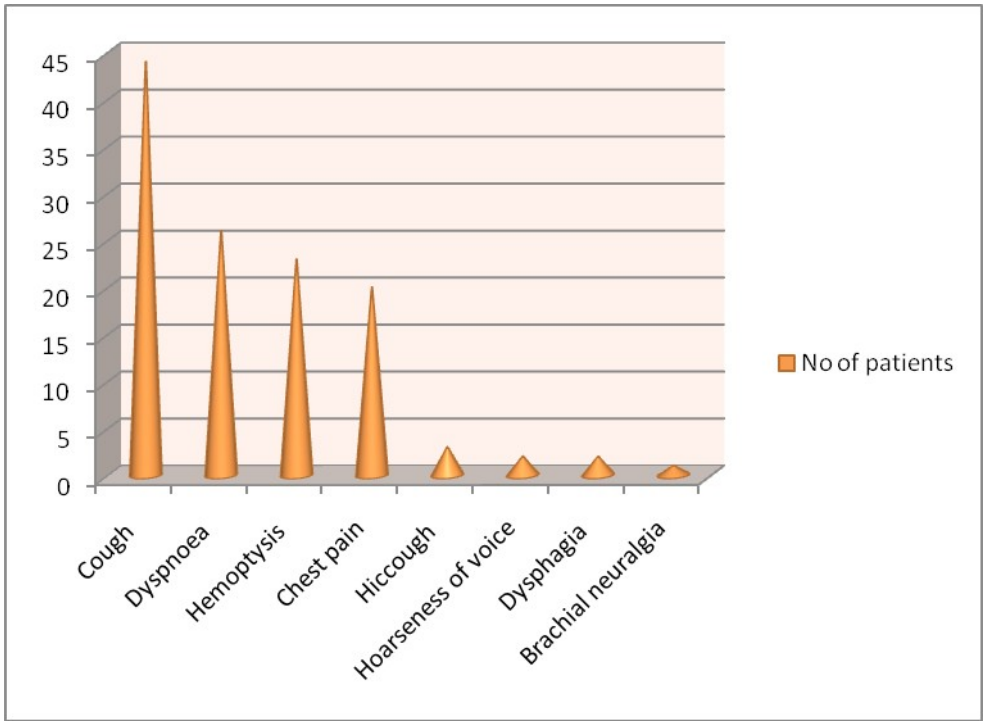
CORRELATION OF PACK YEARS AND BRONCHOGENIC CARCINOMA



Out of 45 patients 26 patients (63%) had pack years of smoking greater than 40 and 15 patients (37%) had pack years less than 40.

In a study conducted by Stefan Diederich, M.D et al[22] in germany, they did ct scan thorax among 700 assymptomatic smokers with pack years > 40, forty percent of individuals had nodules in the lung and 8 smokers had bronchogenic carcinoma. Heavy smoking with pack years > 40 has a definite risk of development of bronchogenic carcinoma, this being substantiated by our study.

SYMPTOM ANALYSIS IN BRONCHOGENIC CARCINOMA



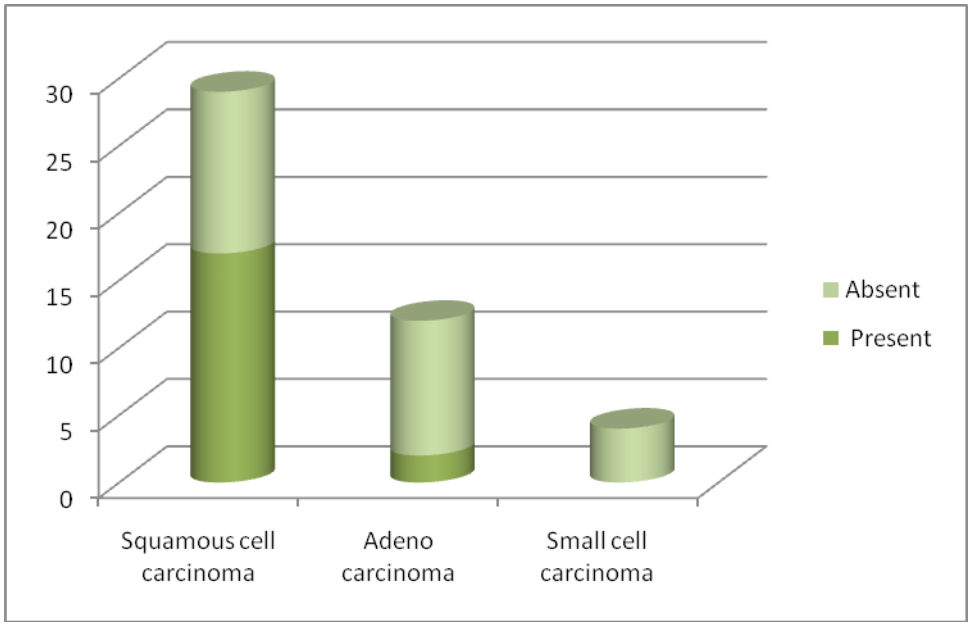
The most common presenting complaint in our study is cough which was present in almost all patients. The next common symptom is dyspnoea which is present in 57 % of patients. Hemoptysis and chest pain were present in 51% and 44% of patients. Hiccough, hoarseness of voice, dysphagia, brachial neuralgia are seen in a small proportion of patients.

In a study conducted by Jindal and Behera 1990[8], the most common symptoms are cough with expectoration, chest pain and hemoptysis, which coincides with our study. In India, where tuberculosis is rampant it is not uncommon to find a lung cancer patient being treated for tuberculosis initially. However, age of the patient, smoking history, mediastinal symptoms such as hoarseness of voice, SVC obstruction and

dysphagia favors the diagnosis of lung cancer.

In our study we couldn't find any correlation between the symptoms and location or pathological type of bronchogenic carcinoma.

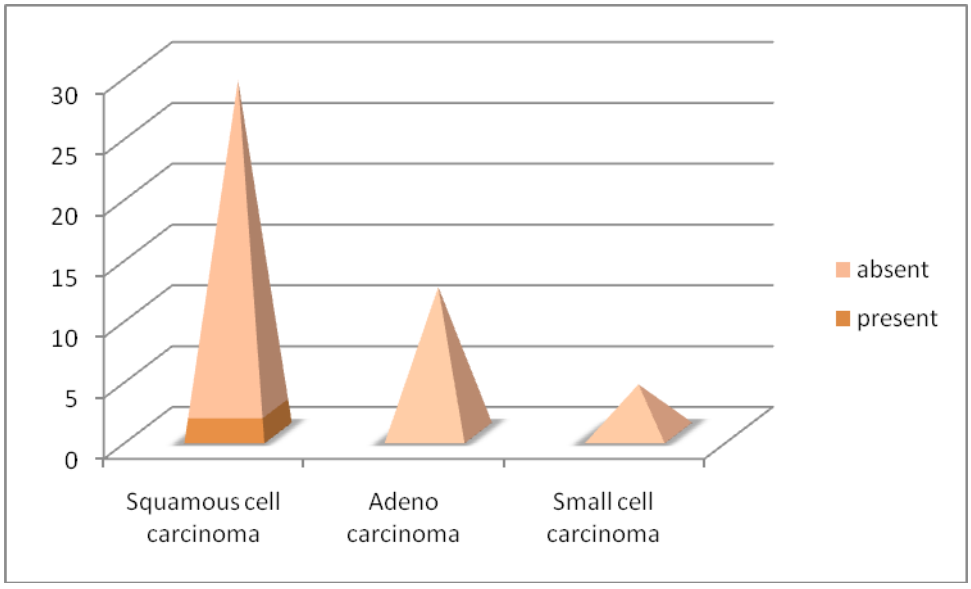
RELATIONSHIP OF CLUBBING WITH PATHOLOGICAL TYPE OF BRONCHOGENIC CARCINOMA



Out of 29 patients with squamous cell carcinoma 17 patients had clubbing. Among 11 patients with adenocarcinoma only 2 patients had clubbing. **The correlation between clubbing and squamous cell carcinoma is found to be statistically significant p value < 0.05 in our study.**

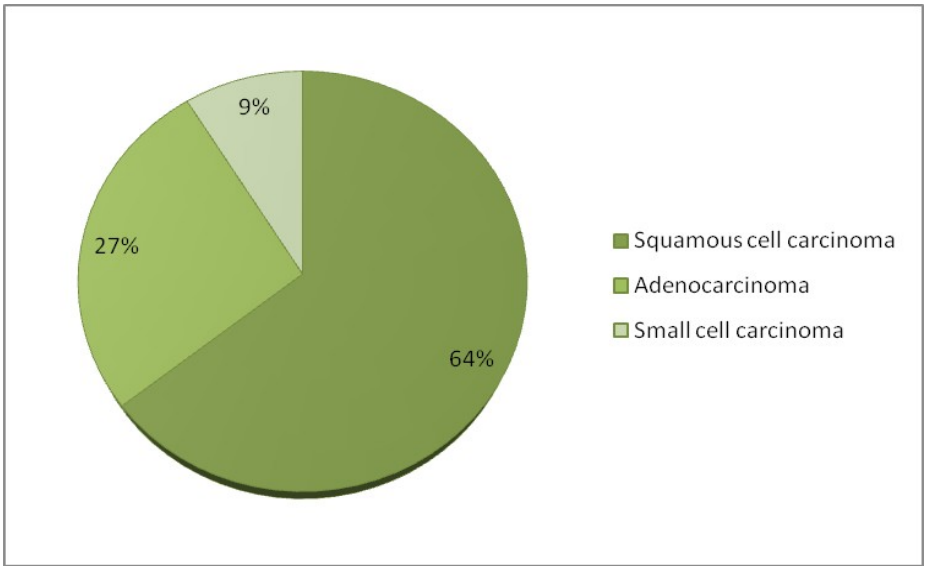
In a study conducted by CM shetty et al in manipal[3], clubbing was present more commonly in squamous cell carcinoma followed by adeno and small cell carcinoma. Eventhough clubbing has been associated with squamous cell carcinoma, there are not much of studies to correlate clubbing with various pathological types of bronchogenic carcinoma. Our study shows a definite correlation between clubbing and squamous cell carcinoma.

RELATIONSHIP BETWEEN SVC OBSTRUCTION AND PATHOLOGICAL TYPE OF BRONCHOGENIC CARCINOMA



Out of 45 patients only 2 patient had SVC obstruction and they both turned out to be squamous cell carcinoma. The most common cause of SVC obstruction is squamous cell carcinoma and small cell carcinoma. Since our study population is less we couldn't derive a statistically relevant correlation between SVC obstruction and bronchogenic carcinoma.

INCIDENCE OF PATHOLOGICAL TYPES OF BRONCHOGENIC CARCINOMA

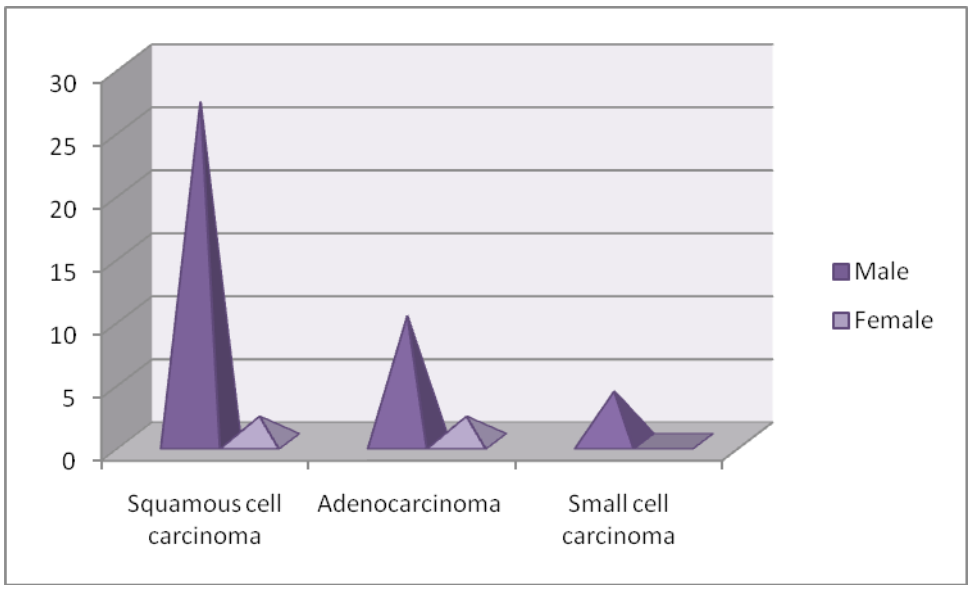


The most common type of bronchogenic carcinoma as per our study is squamous cell carcinoma which constitutes 64% of the total. Adenocarcinoma ranks the second forming 27%, followed by small cell carcinoma cosnstituting 9 percent.

In a study conducted by Kashyap *et al*[16] ,2001 done in northwestern region of india sqaumous cell carcinoma constitutes 58.3%, adeno

carcinoma 10.8% and remaining formed by other cell types. In a study conducted in rajasthan by Gupta, squamous cell carcinoma is the most common pathological type. In a study conducted by Gupta et al[15] 2001 in Chandigarh, the most common malignancy is squamous cell carcinoma forming 60% and adenocarcinoma forming 16.2 percent. The both studies correlates with our study.

RELATIONSHIP BETWEEN PATHOLOGICAL TYPE AND SEX OF THE PATIENT

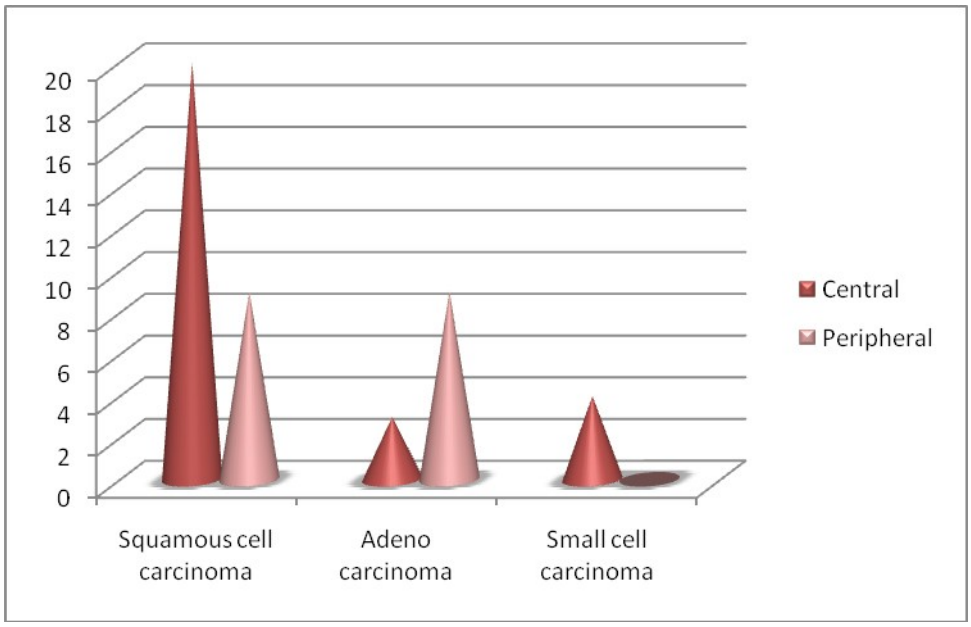


In our study, among 29 patients with squamous cell carcinoma 27 patients were males and 2 were females. Out of 12 patients with adenocarcinoma, 2 patients were females and 10 patients were females.

In a study conducted in pakistan in 2006[2], female sex is associated with adenocarcinoma and male sex with squamous and small cell carcinoma. A prospective study at Sweden found that adenocarcinoma

being the most common type among female population and squamous cell carcinoma in males. In contrast to these studies no definite statistical correlation between adenocarcinoma and females could be made in our study. This could be explained by less number of females affected in our study.

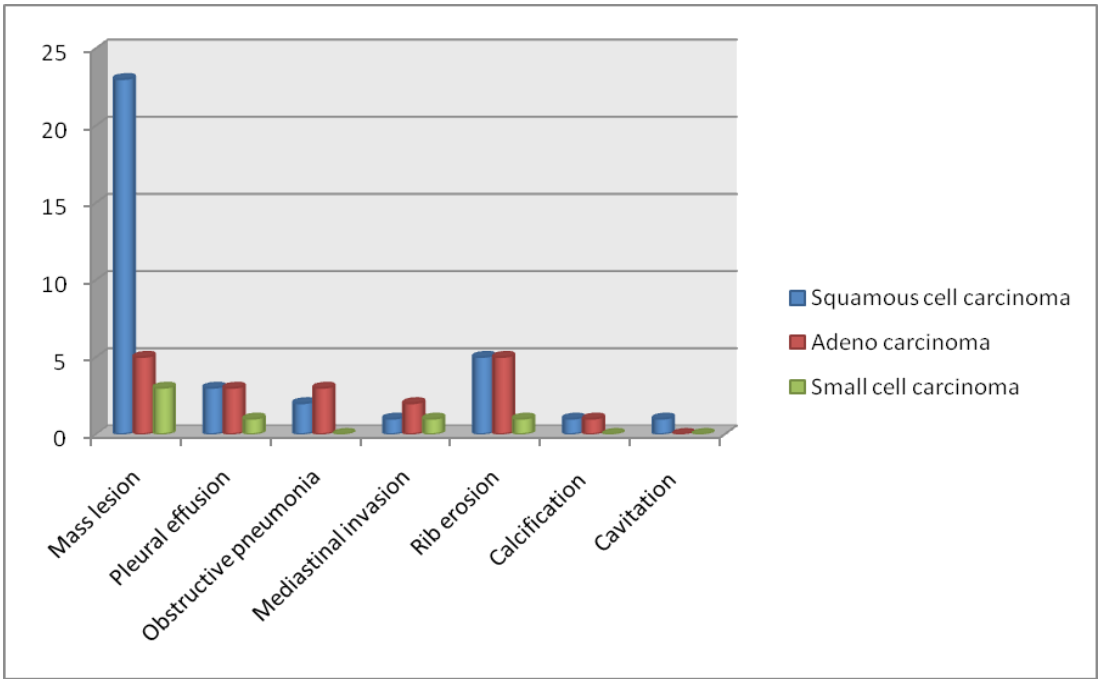
CORRELATION BETWEEN PATHOLOGICAL TYPE AND LOCATION OF BRONCHOGENIC CARCINOMA



Out of 29 patients with squamous cell carcinoma, 20(69%) patients had central tumor and remaining 9(31%) had peripheral tumor. Among 12 patients with adenocarcinoma 9(75%) had peripheral and 3(25%) had centrally placed tumor. The four patients with small cell carcinoma had their lesion located centrally. Chi square was done and t score analysed. P value is <0.05 which is found to be statistically significant.

In an review article presented by D. Behera and T. Balamugesh[7] in February 2004 from PGI Chandigarh who analysed the various studies in bronchogenic carcinoma in india, concluded that Adenocarcinoma presents as a peripheral mass in 61% of cases and in 38.3% as a central lesion. Presentation as a central mass (72.2% cases) is more common among squamous cell carcinoma than as a peripheral lesion (27.8%). Small cell cancer also presents more commonly as a central lesion (83.6%) than as a peripheral lesion (16.4%). This review coincides with our study and **adenocarcinoma presenting as peripheral lesion, squamous cell carcinoma presenting as central lesion was proved to be statistically significant.**

RELATIONSHIP BETWEEN THE RADIOLOGICAL PATTERN AND PATHOLOGICAL TYPE OF BRONCHOGENIC CARCINOMA



The most common radiological presentation in our study is mass lesion with or without collapse of lung. Out of 45 patients, 31 patients(69%) presented as mass lesion in the lung, 7 patients(15%) presented as pleural effusion, 5 patients(11%) presented as obstructive pneumonia.

Among 29 patients with squamous cell carcinoma 23 patients (79%) presented as mass lesion, followed by pleural effusion and obstructive pneumonia. Among 12 patients with adeno carcinoma 5 patients(41%) presented as mass lesion, 3 patients(25%) as pleural

effusion and 3(25%) patients as obstructive pneumonia. In the small cell carcinoma three patients presented as mass lesion and one patient as obstructive pneumonia.

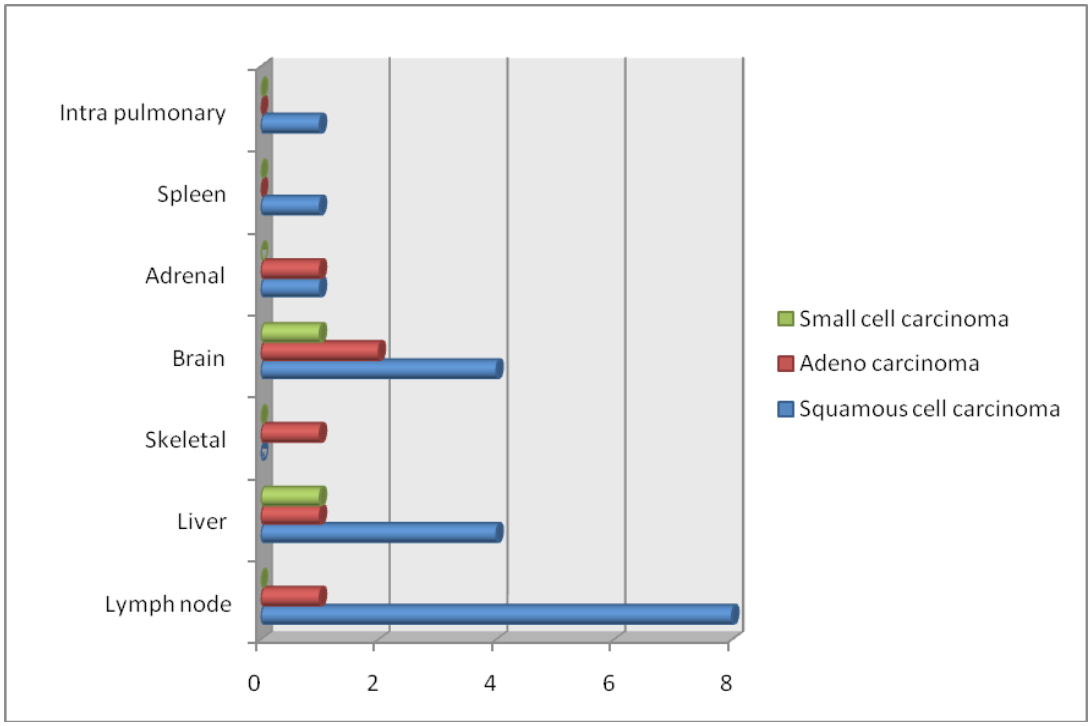
In an review article presented by D. Behera and T. Balamugesh in February 2004[7] from PGI Chandigarh, Mass with or without collapse is the commonest radiological finding in lung cancer in Indian patients. Other findings include pleural effusion in 25.1%, rib erosion and lymphangitis. Isolated pleural effusion has been reported in 3.8% in squamous cell lung cancer, 22% in adenocarcinoma and only 4% in small cell lung cancer. These findings correlates with our study.

In our study, mediastinal invasion is present in 2 patients of adenocarcinoma, one patient of squamous cell carcinoma and small cell carcinoma. Rib erosion was noted in 5 patients with squamous cell carcinoma and 5 patients with adenocarcinoma. Cavitation was seen in one patient with squamous cell carcinoma.

In a study conducted in northern india in Chandigarh[1], mediastinal invasion was present in 19% of patients, rib erosion was present in few patients. This correlates with our study.

INCIDENCE OF METSTASIS IN VARIOUS PATHOLOGICAL

TYPES OF BRONCHOGENIC CARCINOMA



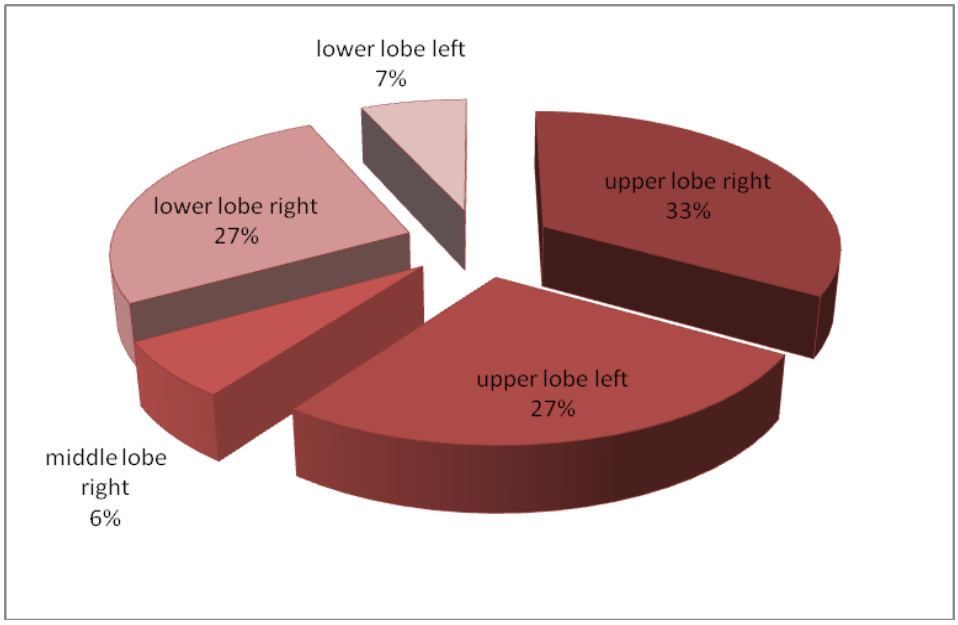
In our study most common site of metastasis is supraclavicular lymph node followed by liver, brain metastasis. The other uncommon sites of metastasis were skeletal, adrenal and spleen. Most of the metastasis were seen with squamous cell carcinoma.

In a study conducted in northern india Chandigarh[1], the commonest site of metastasis were supraclavicular lymph node(26%) followed by liver(10%) and brain(4%).

In a study conducted in korea[4] the most common site of metastasis is supraclavicular lymphnode followed by liver, skeletal and brain metastasis. Both studies correlates with our study.

INCIDENCE OF BRONCHOGENIC CARCINOMA IN VARIOUS

LOBES OF LUNG



Right upper lobe constitutes 33% of brocnchogenic carcinoma, left upper lobe 27%, right lower lobe 27% followed by left lower lobe and right middle lobe. Right side constitutes 66% of the bronchogenic carcinoma. In our study the most common lobe involvement is upper lobe and the most common side involved is right side.

In a study conducted in korea[4] right lung was involved in 60% of patients and left lung was involved in 40% of patients. Upper lobe was predominantly involved when compared to lower lobe involvement.

In an review article presented by D. Behera and T. Balamugesh in February 2004[7] from PGI Chandigarh, Upper zone is involved in most cases followed by mid zone (32.7%), lower zone (16%) and the entire lung (8.8%). These findings correlates with our study.

COMPARISON OF OUR STUDY WITH OTHER INDIAN LARGE

STUDIES

Various studies	Total	Male: female	Age	Smoker: non smoker	Squamous cell carcinoma	Adeno carcinoma	Anaplastic carcinoma
Kashyap et al 2001[16]	638	6.17	54.6	2.4	58.3	-	10.81
Gupta et al 2001[15]	265	7.8	50 to 70	3.6	60	21.5	16.2
Thippanna et al 1998 [13]	160	8.4	40 to 60	4	67.5	8.8	18.7
Rajasekaran et al 1993[11]	232	7.9	53	2.7	72	4.3	3.9
Jindal and Behera 1990[8]	1009	4.5	54.3	2.7	34.3	27.6	25.9
Our study 2008	45	14	59	9.1	64.4	26.6	-

This table shows the comparison of various large studies in india with the present study. The mean age of incidence of bronchogenic carcinoma coincides with the other studies. As in other studies the most common type of bronchogenic carcinoma is squamous cell type followed

by adenocarcinoma and then other types. The incidence of bronchogenic carcinoma in females is much less in the present study when compared to other studies. Smoking: non smoking ratio is more in the present study when compared to other studies. This can be explained by the less incidence of carcinoma in females in our study.

CONCLUSION

In our study of 45 patients,

- The incidence of bronchogenic carcinoma is more common in males compared to females, partly attributed to the low incidence of smoking in south Indian people.
- Highest age incidence is seen in fifth decade of life which is early when compared to other studies is due to Increased smoking, urbanisation, and the introduction of new industries.
- Cigarette smoking is strongly associated with bronchogenic carcinoma. Most of the females are non smokers.
- Bidi smoking is the commonest pattern of smoking habit found which is associated with slightly higher risk of bronchogenic carcinoma when compared to cigarettes.
- All pathological types of bronchogenic carcinoma were associated with smoking.
- Heavy smoking with pack years > 40 has a definite risk of development of bronchogenic carcinoma.
- Cough, hemoptysis and breathlessness were the common complaints in bronchogenic carcinoma.
- Clubbing has a definite correlation with squamous cell carcinoma.
- Adeno carcinoma is associated with peripheral lesion while

squamous and small cell carcinoma is associated with central lesion.

- Squamous cell carcinoma is the most frequent histological type followed by adenocarcinoma.
- The most common radiological pattern found is mass lesion with or without collapse followed by pleural effusion and obstructive pneumonia.
- The most common site of metastasis is supraclavicular lymph node followed by liver, brain metastasis.
- The most common lobe involvement is upper lobe and the most common side involved is right side.

BIBLIOGRAPHY

1) Sk Jindal, Sk Malik, R Dhand, Js Gujral, Ak Malik, Bn Datta, Bronchogenic carcinoma in Northern India. Thorax 1982;37:343-347.

2) Muhammad Imran Suliman, Rushd Jibran, Muhammad Zafar Majeed Demographics of bronchogenic carcinoma patients and frequency of cell types. Gomal Journal of Medical Sciences 2006;4:1-6.

3) Cm Shetty, Bn Lakhar, VSS Ganghadhar, NR Ramachandran. Changing pattern of bronchogenic carcinoma a statistical variation or reality. Ind J Radiol Imag 2005;15:1:233-238.

4) Seung Bong and Byung Sook Choi Observation of primary carcinoma of lung. Younesi medical journal 1964 5:77-79

5) Richarhd. Hoo, Daniel C Campbel. Bronchogenic carcinoma in young people. Chest 1965;48;469-470

6) J. S. M. Leung. Broinchogenic Carcinoma A Surgical Revlew Of 260 Cases. The Bulletin Of The Hong Kong Medical Association 1973 vol 23:36-39.

7) D. Behera and T. Balamugesh. Lung Cancer in India. The Indian Journal of Chest Diseases & Allied Sciences. 2004; 46:269-281.

8) Jindal SK, Behera D. Clinical spectrum of primary lung cancer: Review of Chandigarh experience of 10 years. Lung India 1990; 8 : 94-98.

9) Arora VK, Seetharaman ML, Ramkumar S, et al. Bronchogenic carcinoma: Clinicopathological patten in south Indian population. Lung India1990; 7 : 133-38.

10) Rao S, Rau PVP, Sahoo RC. Bronchogenic carcinoma in the young. Lung India 1992; 10 :101-02.

11) Rajasekaran S, Manickam TG, Vasanthan PJ, et al. Pattern of primary lung cancer: A Madras study. Lung India 1993; 9 : 7-11.

12) Gupta RC, Purohit SD, Sharma MP, Bhardwaj S. Primary bronchogenic carcinoma : Clinical profile of 279 cases from mid-west Rajasthan.Indian J Chest Dis Allied Sci 1998; 40 : 109-16.

13) Thippanna G, Venu K, Gopalkrishnaiah V, Reddy PNS, Sai Charan BG. A profile of lung cancer patients in Hyderabad. J Indian Med Assoc 1999; 97 : 357-59.

14) Arora VK, Sharma V, Reddy KS. Bronchogenic carcinoma in patients below age 40 years and the response to radiotherapy with or withoutCMF regime. Lung India 1998; 16 : 155-58.

15) Gupta D, Boffetta P, Gaborieau V, Jindal SK. Risk factors of lung cancer in Chandigarh, India. Indian J Med Res 2001; 113 : 142-50.

16) Kashyap S, Mohapatra PR, Negi RS. Pattern of primary lung cancer among bidi smokers in North-Western Himalayan region of India. Lung Cancer 2003; 41(Suppl. 2) : S111.

17) Thummalahally Sreeramareddy et al. Clinicopathological profile of primary bronchogenic carcinoma treated in a tertiary care hospital of western Nepal.

18) Ries, L., B. Miller, B. Hankey, C. Kosary, A. Harras, and B. Edwards, editors. 1995. SEER Cancer Statistics Review, 1973-1992: tables and graphs. National Cancer Institute, Bethesda, MD. NIH Publication No. 1995; 96-2789.

19) Auerbach, O., L. Garfinkel, V. Parks, A. Conston, V. Galdi, and L. Joubert. 1984. Histologic type of lung cancer and asbestos exposure. Cancer 54: 3017-3021

20) Dilek Ernam, MD; Figen Atalay, MD; Şükran Atikcan, MD. A Retrospective Evaluation of 571 Lung Carcinoma Patients.2003;4:67-69.

21) Pradad et al Clinicopathological study of bronchogenic carcinoma. Respirology 2004; 9(4):557-560

22) Stefan Diederich et al Screening for asymptomatic early bronchogenic carcinoma with low dose CT of the chest. American Cancer Society International Conference on Prevention and Early

Diagnosis of Lung Cancer, Varese, Italy, 9 December 1998 to 10 December 1998.

23) Vioque J Bolumar F Journal of Epidemiology & Community Health 41(1):74-8, 1987

24) Burton W. Lee, John C. Wain, Karl T. Kelsey, John K. Wiencke, And David C. Christiani . Association of Cigarette Smoking and Asbestos Exposure with Location and Histology of Lung Cancer Am. J. Respir. Crit. Care Med., Volume 157, Number 3, March 1998, 748-755.

25) Viswanathan R, Gupta S, Iyer PVK. Incidence of primary lung cancer in India. Thorax 1962;17 : 73-76.

26) Wig KL, Lazaro EJ, Gadekar NG, Guleria JS.Bronchogenic carcinoma : Clinical features and diagnosis. Indian J Chest Dis 1961; 3 : 209-18.

27) Basu BK, Ghosh TN. A study of bronchogenic carcinoma. Indian J Chest Dis 1971; 13 : 1-9.

28) National Cancer Registry Programme. An epidemiological study. Indian Council of MedicalResearch, Biennial Report 1988-1989, New Delhi.pp. 3-42.

29) Behera D, Kashyap S. Pattern of malignancy in a north Indian hospital. J Indian Med Assoc 1988;86 : 28-29.

30) Sinha BC. Lung cancer: Clinical features. IndianJ Chest Dis 1961; 3 : 209-218.

31) Karai GS, Nath HK, Paul G, Saha D, Roy HK.Carcinoma of the lung : A record and analysis of 100 cases. Indian J Cancer 1967; 4 : 105-13.

32) Shankar PS. Bronchogenic carcinoma. Indian J Chest Dis 1967; 9 : 161-64.

33) Nagrath SP, Hazra DK, Lahiri B, Kishore B,Kumar R. Primary carcinoma of the lung :Clinicopathological study of 35 cases. Indian JChest Dis 1970; 12 : 15-24.

34) Reddy, DB, Prasanthamurthy D, Satyavathi S.Bronchogenic carcinoma : A clinicopathological study. Indian J Chest Dis 1972; 14 :86-89.

35) Guleria JS, Gopinath N, Talwar JR, Bhargava S,Pande JN, Gupta RG. Bronchial carcinoma: An analysis of 120 cases. J Assoc Physicians India1971; 19 : 251-55.

36) Jha VK, Roy DC, Ravindran P. Bronchogenic carcinoma: A clinicopathological study. Indian J Chest Dis 1972; 14 : 78-85.

37) Nafae A, Misra SP, Dhar SN, Shah SNA.Bronchogenic carcinoma in Kashmir valley. Indian J Chest Dis 1973; 15 : 285-95.

38) Malik AK, Aikat BK. Primary pulmonary neoplasm: A histopathologic study. Indian J Cancer 1976; 13 : 149-55.

39) Crofton J, Douglas A. Respiratory diseases. 2nd ed. Oxford: Blackwell Scientific Publications, 1975.

40) Jindal SK, Malik SK, Bansal RC, Banga N. Blood carboxyhaemoglobin in bidi smokers-a preliminary report. Bulletin of the Postgraduate Institute (Chandigarh) 1979;13:222-4.

41) Harrisons principle of internal medicine 17th edition. Mcgraw Hill publications 2007.

42) S.G. Spiro, Oxford Text Book of medicine, 2003. Fourth Edition, 17.14.1-1530.

43) N.A. Khan et al Profile of Lung Cancer The Indian Journal of Chest Diseases & Allied Sciences2006; 48:187-190.

44) Ochsner A, De Bakey M. Carcinoma of the lung. Arch Surg 1941 ; 42:209-8.

45) E L Wynder and J E Muscat. The changing epidemiology of smoking and lung cancer histology. Environ Health Perspect. 1995 November; 103(Suppl 8): 143–148

PROFORMA

Serial No: Occupation: Asbestos exposure

Patients Name: Income:

IP No: Educational Standard:

Age:

Address:

History of presenting illness:

1) Cough Duration:
2) Chest Pain Duration:
3) Hemoptysis
4) Mediastinal Syndrome
5) Superior Venacava Syndrome:
6) Horners Syndrome:
7) Hiccough
8) Hoarseness of voice:
9) Dysphagia

Past History of Pulmonary Tuberculosis-

Smoking Habits:

Type: Cigarette No:of Pack years

Beedi

Pipe smoking

Snuff

Tobacco chewing:

Family history of Bronchogenic Carcinoma

General Examination:

1) Build and Nourishment:
2) Clubbing:
3) Lymphadenopathy:
4) SVC Obstuction:
5) Paraneoplastic Manifestation:
6) Jaundice:

Vital Signs:

Pulse Temp

RR BP

Respiratory System:

1) Shape of Chest:

2) Tracheal Position:

3) Movements:

4) Expansion:

5) Percussion:

6) Breath Sounds:

7) VF, VR

8) Added sounds:

9) Others

Examination of Abdomen: Liver:

Spleen:

Free fluid

Examination of Nervous System:

1) Fundus
2) Horners Syndrome
3) Small muscle wasting in hands

Investigations:

1) Biochemical: Urea, Creatinine, Calcium level, Electrolytes
2) Xray Chest PA, Lateral View:

Location of lesion:

Central

Peripheral

Type of Lesion:

Obstructive Pneumonia

Mass lesion

Pleural Effusion

Mediastinal Invasion

Rib Erosion:

Calcification

Cavitation

3) Sputum Cytology for Malignant cells

4) Bronchoscoy:

Location

BAL lavage

5) CT Scan Thorax

6) Histopathological Diagnosis:

CT guided Biopsy

Bronchoscopic Biopsy

Others- Evidence of metastasis.